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High Prevalence of *Helicobacter pylori* Infection and Histologic Gastritis in Asymptomatic Hispanics

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In this study, we estimated the prevalence of *Helicobacter pylori* infection and histologic gastritis in 58 asymptomatic Hispanic adult volunteers (mean age, 41 years; 59% male) by endoscopic biopsy of the upper gastrointestinal tract. Forty-six subjects (79%) were found to harbor *H. pylori* in gastric biopsies, and all had histologic gastritis. Four other subjects were found to have gastritis in the absence of *H. pylori*. Similar prevalences of *H. pylori* and gastritis were noted in all age groups and also in American-born and immigrant Hispanics. Biopsy data and serologic studies of *H. pylori* antibodies correlated well. We conclude that *H. pylori* infection is an almost universal finding in the gastric mucosa of asymptomatic adult Hispanics, regardless of age. The clinical significance of these findings is unknown, but we speculate that *H. pylori* and its associated gastritis could have a role in the high incidence of gastric carcinoma in Hispanic populations.

Helicobacter pylori (formerly *Campylobacter pylori* [10]) is found commonly in gastric biopsies from symptomatic persons undergoing upper gastrointestinal endoscopy (2–4). The bacterium is believed to be the etiologic agent of chronic nonspecific gastritis (2, 5, 12, 19) and appears to be an important factor in the pathogenesis of duodenal ulcer disease (3, 18). Many studies also have demonstrated (1, 13, 24, 25) that *H. pylori* can be found frequently in asymptomatic, otherwise healthy, volunteers. All such infected persons also have histologic gastritis (1, 13, 24, 25).

In a recent study, we found (6) *H. pylori* in 32% of 113 asymptomatic, predominantly Caucasian, adult volunteers undergoing upper gastrointestinal endoscopy. In this group, *H. pylori* prevalence increased with advancing age (6), reaching a peak of 48% in those aged 60 to 69 years. Conversely, data from serological studies in developing nations have demonstrated prevalence rates of 80 to 90% for *H. pylori* infection in all adults, regardless of age (17, 20, 23). We wondered, therefore, if differences in the prevalence of *H. pylori* infection exist between racial groups within the United States. In this study, we estimated the prevalences of *H. pylori* infection and histologic gastritis in a group of Hispanic volunteers residing in the Los Angeles area.

MATERIALS AND METHODS

Subjects. Asymptomatic healthy Hispanic adults were recruited from the general population by means of advertisements placed in Spanish-language newspapers. As described previously (6), prospective candidates were carefully screened to ensure that they were truly asymptomatic. A total of 45 subjects were enrolled in the study. An additional 13 Hispanic subjects had participated in our initial studies (6), and these individuals are also included in this report. All participants in the study gave written informed consent under a protocol approved by the Research Committee of the Los Angeles County-University of Southern California Medical Center. The mean age of the subjects was 41 years (range, 18 to 81), and 59% were male. None of the elderly subjects was institutionalized.

Gastrointestinal endoscopy. All subjects underwent endoscopy and biopsy of the upper gastrointestinal tract. Before the endoscopy, 5 to 8 ml of venous blood was withdrawn, and the resultant serum was stored at −4°C. During the endoscopy, paired biopsy samples were obtained routinely from the duodenal bulb, the prepyloric gastric antrum, the gastric corpus, and the distal esophagus (approximately 5 cm above the squamocolumnar junction). Separate sterile biopsy forceps were used for each site. One biopsy specimen from each pair was placed in transport medium and submitted for microbiological assessment. The remaining biopsy specimen was fixed in neutral buffered formalin and retained for histologic study. Incidental lesions found during endoscopy also were biopsied for histologic evaluation.

Microbiological techniques. Immediately after endoscopy, the biopsy specimens were removed from the transport medium, plated onto both chocolate and *Campylobacter*-selective agars (Scott Laboratories, Los Angeles, Calif.), and cultured at 37°C under microaerobic conditions (Campypak; BBL Microbiology Systems, Cockeysville, Md.). The plates were checked every 2 to 3 days, and identification of *H. pylori* was made on the basis of the morphological features of the colony and Gram stain and positive reactions to oxidase, catalase, and urease.

Histologic studies. Gastric biopsy specimens were stained with hematoxylin and eosin and graded for gastritis on the basis of the inflammatory cell infiltrate as described in previously published criteria (6). Chronic gastritis was denoted by a lymphocytic infiltrate, and active chronic gastritis was denoted by a neutrophilic infiltrate in addition to the finding of chronic gastritis. In addition to inflammation, glandular atrophy and intestinal metaplasia were also noted when present.

The identification of *H. pylori* in all specimens was made by reviewing the hematoxylin and eosin stains (6). The histologic evaluation of the duodenal biopsies was based on our previously published criteria (9), and that of the esoph-

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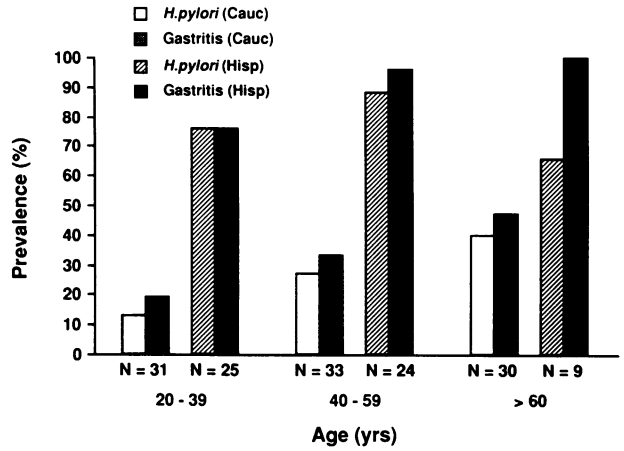

FIG. 1. Prevalence of *H. pylori* infection and histologic gastritis, as determined by gastric biopsy, in asymptomatic adult Hispanics (Hisp) grouped by age. Data from asymptomatic adult Causcasians (Cauc) are provided for comparison (derived from Dooley et al. [6]).

ageal biopsy specimens was based on the criteria of Ismail-Beigi et al. (16).

Serologic technique. Serum samples were examined for *H. pylori* by an enzyme-linked immunosorbent assay described elsewhere (6, 22). In brief, the antigen used was a pool of the sonicated isolates of *H. pylori* from five patients with gastritis. Serum samples were diluted 1:50 for the immunoglobulin (Ig) A assay and 1:800 for the IgG assay. The second antibody was peroxidase-conjugated goat anti-human IgA or IgG. All samples were coded and examined without knowledge of culture and histologic data.

Analysis of data. A subject was considered to be infected with *H. pylori* only if the bacterium was cultured from or visualized in biopsy specimens.

RESULTS

Endoscopy. Endoscopy revealed unsuspected lesions in 11 of the 58 subjects, mucosal erosions in 9, and subepithelial hemorrhages in 2. Only two of the nine subjects with erosions were using nonsteroidal anti-inflammatory drugs, while both subjects with subepithelial hemorrhages were using these agents. Mucosal ulceration was not found in any subject.

Prevalence of *H. pylori* and gastritis. Forty-six of the 58 volunteers (79%) were found to have *H. pylori* on gastric biopsies. Of these 46, culture and histologic stain were both positive in 38, culture only was positive in 3 (sensitivity, 89%), and stain only was positive in 5 (sensitivity, 93.5%). In contrast to our prior findings in Caucasians, similar prevalence rates were noted in all Hispanic age groups (Fig. 1). Prevalence rates for *H. pylori* were similar in immigrant and American-born Hispanics (29 of 35 subjects versus 17 of 23 subjects).

Gastritis was observed in 50 subjects (86%). All 46 with *H. pylori* on gastric biopsies had gastritis. Gastric biopsies were read as normal in only seven subjects. One additional subject was noted to have glandular atrophy of the corpus without concomitant inflammation. As noted for *H. pylori*, prevalence rates for gastritis were similar in all age groups (Fig. 1).

Duodenal findings. Adequate duodenal biopsies were available in 55 individuals. *H. pylori* was cultured from duodenal biopsies in 10 subjects (18%), and all 10 had *H. pylori* in gastric biopsies. In only one of these 10 subjects was *H. pylori* observed in hematoxylin-and-eosin-stained duodenal specimens. Interestingly, this subject also had gastric metaplasia of the duodenum. Gastric metaplasia of the duodenum was noted only in three subjects (5%).

TABLE 1. Serologic results

Biopsy data[a]	Total	No.			
		IgA		IgG	
		+	−	+	−
Gastritis +/*H. pylori* +	44[b]	27	17	40	4
Gastritis −/*H. pylori* −	7	0	7	0	7
Gastritis +/*H. pylori* −	4	3	1	3	1
Corpus atrophy/*H. pylori* −	1	1	0	1	0

[a] +, positive; −, negative.
[b] Serum was unavailable in two subjects (both were *H. pylori* positive on biopsy).

Twenty-seven subjects were found to have normal duodenal histology. An additional 23 subjects were diagnosed as having grade 1 duodenitis, characterized by a mild increase in mononuclear cells, which is not believed to be of pathological significance (9). Only five subjects were deemed to have significant duodenitis, and, of these, four showed an increase in mononuclear cell infiltrate only. All four of these individuals had *H. pylori* on gastric biopsies. The remaining subject had intramucosal neutrophils in addition to an increased mononuclear cell infiltrate. This was the subject who had *H. pylori* in association with gastric metaplasia of the duodenum.

Esophageal findings. *H. pylori* was cultured from esophageal biopsies in five subjects, all of whom had *H. pylori* on biopsies of the gastric corpus. Histologically, *H. pylori* was not visualized in esophageal biopsies and no abnormalities of esophageal histology were noted.

Comparative findings of the gastric antrum and corpus. Forty of the 46 individuals with *H. pylori* infection were found to have the bacterium in biopsies from both the gastric antrum and corpus. *H. pylori* was noted in antral biopsies only in three subjects and in corpus biopsies only in three subjects. Gastritis was found in biopsies from both the antrum and the corpus in 37 of the infected individuals. Five infected subjects had gastritis of the antrum only, while four had gastritis of the corpus only. Active chronic gastritis was observed only in subjects with *H. pylori* and was more common in the antrum than the corpus (18 versus 13 subjects).

Four subjects were found to have gastritis in the absence of *H. pylori* on biopsy. Of these four, two had gastritis of both the antrum and the corpus, one had gastritis of the corpus only, and the remaining subject had gastritis of the antrum and glandular atrophy of the corpus. As noted above, there was one other subject who had glandular atrophy of the corpus only.

Serologic results. IgG antibody to *H. pylori* was detected in 91% of subjects who had the bacterium on gastric biopsy, while IgA antibody was detected in 61% (Table 1). All seven subjects who had neither gastritis nor *H. pylori* upon gastric biopsy were found not to have antibodies directed against the bacterium (Table 1). Three of the four subjects with *H. pylori*-negative gastritis were found to have both IgA and IgG antibodies to the bacterium (Table 1). The person without an antibody response was the individual who had gastritis of the antrum and atrophy of the corpus (see above).

Lastly, the subject who had glandular atrophy of the corpus only was noted to have IgG and IgA antibodies directed against *H. pylori*.

DISCUSSION

This study demonstrates that *H. pylori* infection is very common in asymptomatic Hispanic subjects residing in the Los Angeles area. This was not unexpected as preliminary data have shown a similarly high prevalence of the bacterium in young Colombian adults (14) and Peruvian children (17). It is interesting that prevalence rates were similar in both immigrant and American-born Hispanics. Therefore, marked variations in the prevalence of *H. pylori* exist between Caucasians and Hispanics residing in the Los Angeles area (Fig. 1) (6). The prevalence of *H. pylori* is low among young Caucasian adults and increases steadily throughout adult life (6). It is estimated that the incidence of *H. pylori* infection is between 1 and 2% per annum in U.S. Caucasian adults (21). In Hispanics, however, young adults demonstrate a very high prevalence rate of the bacterium and this changes little throughout adult life, consistent with acquisition of infection during childhood and adolescence (17). Preliminary data suggest (11) that the prevalence of *H. pylori* is increased in Afro-Americans. A more recent study confirms these findings (15). Therefore, studies examining the role of *H. pylori* in gastroduodenal diseases must control for these ethnic differences.

The factors responsible for the differences in prevalence rates of *H. pylori* between different nations (6, 14, 17, 20, 21, 23) and between different ethnic groups within the same nation (11, 15) are unknown. Furthermore, the precise mechanisms responsible for the transmission of *H. pylori* have not been defined, although available data favor person-to-person transmission (7). *H. pylori* binds specifically to certain sites on the surface of the gastric epithelial cell (8). It is possible that the presence of, and the affinity for, these binding sites may be genetically determined. Preliminary evidence suggests (17), however, that the higher prevalence rates of the infection in certain ethnic groups correlate inversely with socioeconomic status. However, data suggesting that socioeconomic status is an important factor do not preclude the possibility that genetic factors may also play a role.

The data from this and our previous studies (6) confirm that *H. pylori* is associated with histologic gastritis in asymptomatic individuals as it is in symptomatic patients (1, 13, 24, 25). *H. pylori* is believed to be the etiologic agent of this gastritis (5–7, 12, 19) and cannot be considered to be a simple commensal of the human stomach. The significance of this finding in asymptomatic, otherwise healthy, individuals is unknown. The presence of *H. pylori* itself cannot be accepted as a cause of upper gastrointestinal symptoms, since the majority of infected individuals remain symptom free. However, extensive studies in Scandinavia (26) and Colombia (4) have demonstrated that histologic gastritis is a precursor lesion in the development of gastric carcinoma. The incidence of gastric carcinoma is higher in these nations than in the United States, and it is possible that infection with *H. pylori* in childhood might act as an initiating event in the development of gastric carcinoma.

This study confirms a number of findings from our previous study (6). *H. pylori* and inflammation are found in both the antrum and the corpus in the majority of infected individuals. Furthermore, active chronic gastritis is found only in infected subjects and is more common in the antrum than the corpus. A small number of subjects have gastritis in the absence of *H. pylori* on gastric biopsy. Some of these individuals are noted to have an antibody response to *H. pylori*. It is possible that biopsies failed to detect *H. pylori* in these subjects. Alternatively, the subjects might have cleared the bacterium spontaneously, while the antibody response persisted.

This study also confirms that significant duodenitis is unusual in asymptomatic individuals (9). *H. pylori* may be cultured frequently from duodenal biopsies in asymptomatic subjects (9), but histologic visualization of the bacterium in these biopsies is rare. In contrast, among patients with duodenal ulceration (18), *H. pylori* is associated with gastric metaplasia of the duodenum in more than 50% of cases. Culturing the bacterium from duodenal biopsies, in the absence of direct visualization in stained biopsy specimens, may be the result of contamination of the biopsies by gastric contents. In our previous study, we observed (9) gastric metaplasia in duodenal biopsies from 22% of asymptomatic individuals. In the current study, however, gastric metaplasia was seen in only 5% of asymptomatic Hispanics. The significance of this difference is unknown.

In summary, this study shows that *H. pylori* is highly prevalent in asymptomatic adult Hispanics. The bacterium is associated with gastritis in all cases, adding further evidence to the contention that *H. pylori* is the cause of the histologic lesion. We conclude that the presence of the bacterium and the associated gastritis is not a sufficient factor to cause symptoms.

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